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Atrial Premature Depolarization-Induced Changes in QRS and T Wave Morphology on Resting Electrocardiograms in Horses

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Background: The electrocardiographic differentiation between atrial (APDs) and ventricular (VPDs) premature depolarizations is important. P wave prematurity and normal QRS and T wave morphology generally are used as discriminating criteria for APDs.

Hypothesis/Objectives: The aim of this study was to determine whether P, Q, R, S, and T wave amplitude, PQ interval, QRS and P wave duration and P and T wave morphology differ between APDs and sinus beats. To determine the relationship between the RR coupling interval and the change in S wave amplitude between sinus beats and APDs.

Methods: Case–control study. From a modified base-apex configuration of 30 horses with APDs at rest, sinus beat and APD associated preceding RR interval, P, PQ and QRS duration and P, R, S, and T wave amplitudes were measured. Linear mixed models and logistic regression were used to determine the effect of APDs on the ECG variables studied.

Results: In comparison to sinus beats, APDs were associated with a significant ($P < .001$) change in P amplitude (-0.03 ± 0.01 mV) and increase in S (0.20 ± 0.02 mV) and T (0.08 ± 0.03 mV) amplitude. PQ (-20.3 ± 5.2 ms) and RR (-519 ± 14 ms) interval and P duration (-21.1 ± 3.0 ms) decreased ($P < .001$). APDs were significantly associated with a singular positive P wave (OR: 11.0, $P < .001$) and were more likely to have a monophasic positive T wave (OR: 9.2, $P < .001$). A smaller RR coupling interval was associated with an increased relative difference in S amplitude ($P < .01$).

Conclusions: Atrial premature depolarizations may lead to changes in QRS and T wave morphology. Knowledge of these changes is important to avoid interpreting certain APDs as VPDs.

Key words: Cardiology; Electrocardiography; Equine.

Electrocardiography is of increasing importance in equine medicine. Correct classification of different electrocardiographic complexes is important to differentiate clinically relevant abnormalities from physiological complexes. Electrocardiographic differentiation between ventricular (VPDs) and atrial premature depolarizations (APDs) is important because the clinical relevance of both types of premature cardiac complexes is very different. During exercise, VPDs, especially when consecutive, are thought to be a risk factor for the induction of ventricular tachyarrhythmia, which can cause an abrupt decrease in blood pressure and may even lead to cardiovascular collapse. Atrial premature depolarizations can occur in normal horses but do, however, predispose to the development of atrial fibrillation.[1,2] Because management and treatment options can vary depending on the diagnosis, it is important to correctly differentiate between APDs and VPDs.

Abbreviations:

APD	atrial premature depolarization
SD	standard deviation
TVEC	transvenous electrical cardioversion
VPD	ventricular premature depolarization

Ventricular dysrhythmias are the consequence of abnormal impulses arising somewhere in the ventricular myocardium, leading to a different ventricular conduction, and resulting in changes in QRS and T wave morphology and duration.[1–3] However, depending on the site of origin, changes in ventricular conduction and morphology can sometimes be minimal.[2] Ventricular premature depolarizations arise from the ventricular myocardium and result in AV dissociation: they are not associated with a preceding P wave and the normal sinus P wave often is not conducted, resulting in a compensatory pause.[4] Atrial dysrhythmias are caused by abnormal impulse formation in the atrial myocardium outside of the sinoatrial node. In the case of APDs, ventricular conduction is not affected and QRS complex and T wave morphology remain unchanged.[1,3,5] A premature P wave, with normal or abnormal morphology, usually is present, but can sometimes be buried in the preceding QRS or T wave and thus be difficult to visualize. If a P wave is not clearly visible, differentiation between a VPD and an APD can be challenging, especially when there are only mild changes in QRS and T wave morphology. Using multiple lead recordings can

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be helpful.[6,7] Each lead measures the cardiac depolarization from a different angle, so minor electrical changes sometimes can be more easily identified in 1 lead compared to the other.

The aim of our study was to report P, Q, R, S, and T wave morphology associated with APDs at rest to improve the interpretation of electrocardiogram recordings. Therefore, the amplitude of P, Q, R, S, and T waves and the PQ interval and P and QRS duration of APDs and normal sinus beats were compared. Associations between APDs and specific T and P wave morphologies were investigated. Finally, the effect of the RR interval on the relative change in S wave amplitude following an APD was studied.

Material and Methods

Study Design and Population

A case–control study was performed on 30 ECGs of 30 horses with APDs. Per horse, 10 single APDs were included, whereby for each APD the preceding sinus beat was taken as its negative control. All ECGs belonged to horses presented to the Departement of Large Animal Internal Medicine, Faculty of Veterinary Medicine, Ghent University for cardiac examination. All data were used with owner informed consent. Mean age and body weight of the horses was 11 ± 5 years and 569 ± 81 kg, respectively. Two stallions, 18 geldings and 10 mares were included. Breed was reflective of the hospital population, with 26 warmblood horses, 2 Arabian horses, 1 French trotter, and 1 Belgian draft horse. Nineteen horses had been presented for cardiac examination after electrical cardioversion (TVEC) of atrial fibrillation (4 days [n = 14] and 6 weeks [n = 5] after TVEC). Two horses were diagnosed with mild to moderate mitral regurgitation, 1 horse had an aortacardiac fistula, and 1 horse had suffered from intoxication with cardiac glycosides. Seven horses had APDs without any other cardiac abnormalities. Horses with VPDs were excluded from the study.

Electrocardiography

In all 30 horses, an ECG was recorded using a Televet100 recording system.[a] Four self-adhesive electrodes were placed underneath a girth.[b] The right arm electrode was positioned 15 cm right of the withers, the left leg electrode caudal to the left elbow on the thorax and the left-arm electrode 10 cm above the left leg electrode, resulting in a modified base-apex configuration.[6,8] A reference electrode was placed on the left side of the withers. All electrodes were connected to the recording device, placed in the girth. The ECG recordings were analyzed offline using dedicated software.[c] Standard gain (10 mm/mV) and paper speed (25 mm/s) were increased to 20 mm/mV and 200 mm/s to allow accurate analysis. Three different leads were displayed simultaneously to improve ECG interpretation. (Fig 1). In the Televet system, leads 1 and 2 are recorded, whereas lead 3 is calculated, assuming an Einthoven triangle. Measurements always were performed in lead 2 [between the right arm (−) and left leg (+) electrode]. For each horse, the first 10 APDs and their preceding normal sinus beats were included. An APD was defined as an individual atrial depolarization, occurring prematurely, meaning with a preceding RR coupling interval at least 20% (arbitrary value) shorter than the RR interval of the preceding sinus beat. Only APDs followed by a QRS complex were included in the study. The ECGs were recorded at rest and heart rate varied between 35 and 55 beats/min. For both the sinus beats and the APDs, preceding RR interval, P, PQ and QRS duration and P (P1 and P2 if present), Q, R, S, and T (T1 and T2 if present) wave amplitudes were measured (Fig 2).

Although measurements always were performed in lead 2 of the ECG, 3 different leads were recorded to allow for correct interpretation of the electrocardiogram (Fig 1). When P waves were difficult to visualize in lead 2 of the ECG, they often were better visualized in lead 1 or lead 3, so that a correct diagnosis of APD could be made.

Data Management and Statistical Analysis

Statistical analyses were performed using SAS 9.4.[d] In Fig 2, the different measurements applied are shown. If the P wave was bifid or biphasic, only the largest amplitude was selected. Singular, bifid, or biphasic nature of the wave was recorded as a categorical variable. For the T wave, if biphasic, the sum of the positive amplitude and the absolute value of the negative amplitude were used for statistical analysis. The T wave morphology was recorded as a categorical variable with 4 categories: biphasic positive-negative, biphasic negative-positive, monophasic positive, and monophasic negative.

To determine the effect of an APD on P, Q, R, S, and T amplitude and P, PQ and QRS duration, a linear mixed model was constructed with horse added as a random factor to account for clustering of measurements within a horse (PROC MIXED). A maximum likelihood model with Satterthwaite approximation was used. The outcome variables were tested for normal distribution (histograms and Q–Q plots) and validity of the final models was checked by inspecting the residuals.

Logistic regression was performed to determine associations between P and T wave morphology and APDs and RR intervals,. A generalized linear model (PROC GLIMMIX) was used with binomial distribution and logit link function with Wald's statistics for type 3 contrasts. Horse was added as a random factor to account for the clustering of ECG recordings within a horse. Post-hoc tests were done with Bonferroni corrections for multiple comparisons. Model fit was evaluated by the Hosmer-Lemeshow goodness-of-fit test for logistic models. In all models, significance was set at $P < .05$.

A mixed model approach (PROC MIXED) was applied to determine the relationship between the RR coupling interval of APDs and the change in S wave amplitude. The percentage change in S wave amplitude between a normal sinus beat and an APD was calculated as follows: S wave amplitude (sinus beat)/S wave amplitude (APD) × 100. A maximum likelihood model with Satterthwaite approximation was used, and horse was added as a random effect. The outcome variables were tested for a normal distribution (histograms and Q–Q plots) and validity of the final models was checked by inspecting the residuals. In addition to the general analysis, to better understand the horse effect, the relationship between the RR coupling interval and the change in S wave amplitude was visually inspected and analyzed by simple linear regression in every individual horse. Validity of the models was checked by inspecting the residuals.

Results

Changes in ECG Measurements

In total, 588 complexes (294 APDs and 294 preceding sinus beat controls) from 30 horses were collected. In 2 horses, only 6 and 8 APDs and preceding sinus beats could be measured, respectively. In all other horses (n = 28) 10 APDs and their preceding sinus beats were included. For 96 of 300 APDs (32%), the ectopic P wave could not be accurately measured because it was

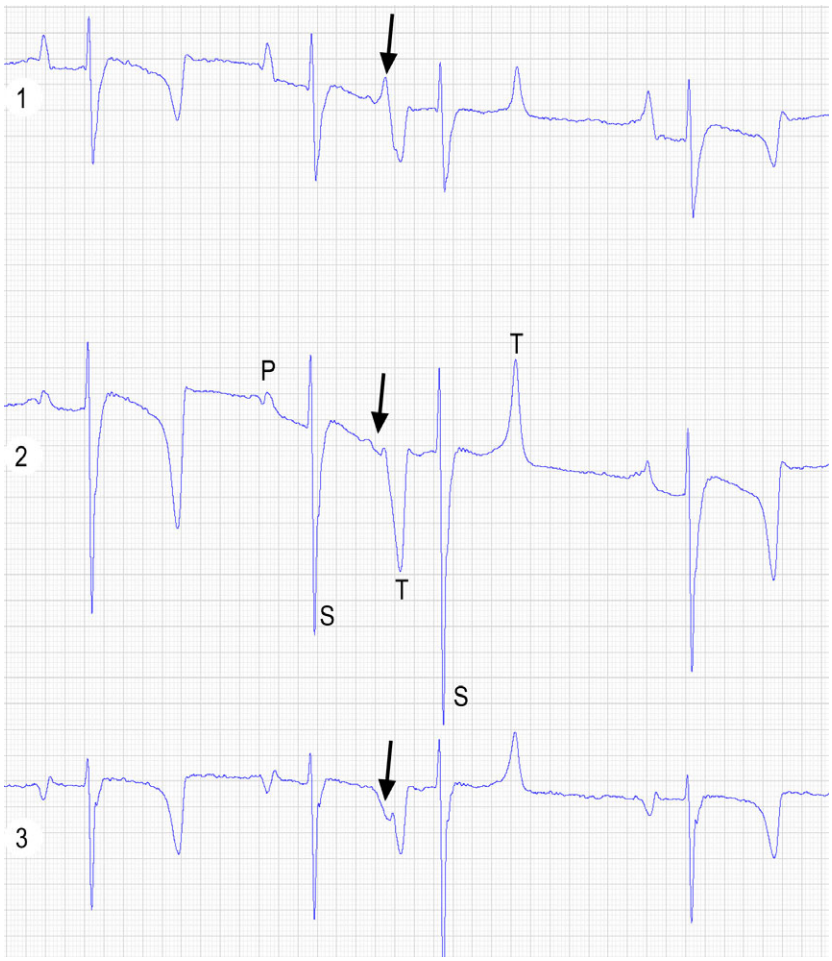

Fig 1. Electrocardiogram with 3 lead recording (1–3) showing an atrial premature depolarization (APD). In lead 2 the P wave of the APD is partially buried in the preceding T wave and difficult to identify. The S wave amplitude after the APD is increased and also T wave morphology has changed. Lead 1 and 3 clearly show the premature P wave. Gain: 20 mm/mV, paper speed: 100 mm/s.

fully or partially buried in the preceding T wave. In all horses, the majority of the QRS complexes had an rS morphology, in 6 horses an S morphology was present in 5–35% of the complexes per recording. A Q wave was not visible in any of the horses. The S and the T amplitudes of APDs were significantly ($P < .001$) increased as compared to sinus beats (mean difference ± SD 0.20 ± 0.02 and 0.08 ± 0.03 mV, respectively) and PQ (−20.3 ± 5.2 ms) and RR (−519 ± 14 ms) intervals were decreased as compared to sinus beats ($P < .001$). Furthermore, the P wave of an APD significantly differed from the P wave of a regular sinus beat (amplitude −0.03 ± 0.01 mV, duration −21.1 ± 3.0 ms). The R amplitude ($P = .51$) and QRS duration ($P = .09$) were not significantly different between APDs and sinus beats (Table 1).

The change in morphology was significantly horse dependent for all variables, especially for the S wave amplitude ($P < .0001$). In some horses, there was a large increase in the S wave amplitude in case of an APD whereas in other horses S wave amplitude hardly changed. The horses with the highest and lowest change in S wave amplitude had a mean±SD increase in S amplitude of 44 ± 10 and 3 ± 5%, respectively.

Changes in P and T Wave Morphology

To determine associations between P wave and T wave morphology and APDs, 492 P waves (40% APDs, 60% sinus beats) and 588 T waves (50% APDs, 50% sinus beats) could be used. The APDs were significantly associated with a singular positive P wave (OR: 11.0, $P < .001$) and were respectively more and less likely to have single positive (OR: 9.2, $P < .001$) or single negative (OR: 0.2, $P < .001$) T waves (Table 2). The RR interval significantly influenced T wave morphology ($P < .05$). Within APCs, a monophasic positive T wave was associated with a shorter RR interval (mean ± SD: 935 ± 46 ms), whereas monophasic negative T waves were associated with the longest mean RR interval (1,485 ± 62 ms). Those APCs with biphasic negative-positive T waves had a mean RR interval of 1,286 ± 42 ms. In normal sinus beats, the P wave was most often bifid (79.6%), whereas 62.2% of APDs had a

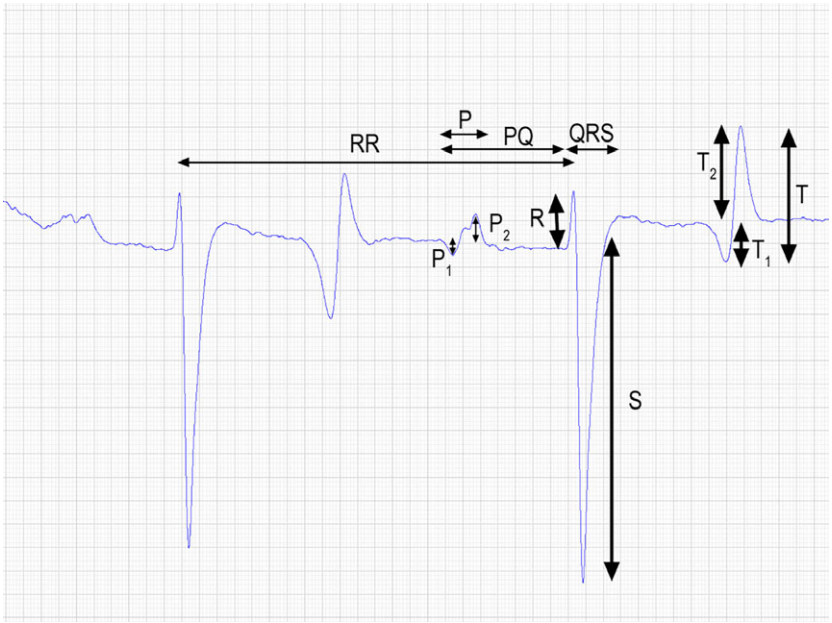

Fig 2. Method of measurement of the different electrocardiographicvariables used in this study. For all atrial premature depolarizations and their preceding sinus beat RR and PQ interval, QRS duration and P1, P2, Q, R, S, and T1 and T2 wave amplitude were measured. If a P wave was biphasic or bifid, only the largest amplitude was included in statistical analysis. For biphasic T waves, the total T wave amplitude (T), calculated as the sum of the absolute amplitudes of the positive and negative T wave (T1, T2) was used for statistical analysis.

Table 1. Changes in ECG morphology in 588 recordings of 30 horses with APDs.

Variable	n	Sinus Beat Mean ± SD (Range)	APD Mean ± SD (Range)	Estimated Difference	95% CI of Difference	*P*-value
P amplitude (mV)	492	0.25 ± 0.08 (0.05–0.60)	0.22 ± 0.11 (0.05–0.70)	−0.03 ± 0.01	−0.04 to −0.02	<.001
R amplitude (mV)	574	0.27 ± 0.24 (0.00–1.00)	0.26 ± 0.21 (0.00–1.00)	−0.01 ± 0.01	−0.04 to 0.02	.51
S amplitude (mV)	588	−2.16 ± 0.54 (−4.20 to −0.90)	−2.35 ± 0.61 (−4.10 to −1.00)	−0.20 ± 0.02	−0.24 to −0.16	<.001
T amplitude (mV)	588	0.97 ± 0.05 (−2.10 to 1.10)	0.90 ± 0.05 (0.30–1.80)	0.08 ± 0.03	0.04 to 0.11	<.001
P duration (ms)	477	168 ± 42 (56–272)	147 ± 45 (60–254)	−21.1 ± 3.0	−26.95 to 15.3	<.001
PQ interval (ms)	495	337 ± 74 (148–748)	321 ± 97 (0–633)	−20.3 ± 5.2	−30.4 to −10.2	<.001
RR interval (ms)	588	1,468 ± 327 (566–2,320)	1,002 ± 225 (522–2,000)	−519 ± 14	−546 to −492	<.001
QRS duration (ms)	588	132 ± 12 (98–164)	133 ± 11 (98–162)	1.0 ± 0.6	−2.1 to −0.2	.09

APD, atrial premature depolarization; CI, confidence interval; SD, standard deviation.
Horse was added as a random factor and was significant in each model ($P < .001$).

monophasic positive P wave. As for the T wave, in normal sinus beats it was most often biphasic negative-positive (67.3%), with a fairly small percentage monophasic positive (16.7%) or monophasic negative (15.3%) T waves. In APDs, however, almost half of the T waves changed polarity toward a monophasic positive morphology (41.5%; Table 2). The horse effect was significant ($P < .001$) for P wave morphology, but not for T wave morphology.

Relationship between RR Coupling Interval and S Wave Amplitude

A total of 294 observations could be used to study the relationship between the RR interval and S wave amplitude. A significant negative relationship between RR coupling interval of the APD and the relative difference in S wave amplitude between a sinus beat and an APD was detected ($P < .01$). The random horse effect was significant ($P < .001$), and therefore detailed observations were made in every horse. In only 13% (4/30) of the horses, there was a significant ($P < .05$) negative relationship between the RR coupling interval and the difference in S wave amplitude, and in 1 horse a significant positive relationship (P < .01) was found. In the other horses, there was no significant relationship.

Discussion

Our study shows that, besides a change in P wave morphology, APDs can induce changes in QRS complex and T wave morphology. In APDs, the S and T wave amplitude increased and PQ and RR interval decreased, whereas R amplitude and QRS duration did

Table 2. Changes in P and T wave morphology associated with APDs.

	Sinus beat (%)	APD (%)	OR	95% CI	*P*-value
P wave					
Bifid	223 (79.6)	60 (30.0)	Ref.		
Singular	66 (22.7)	125 (62.2)	11.0	6.1–20.0	<.001
Biphasic	2 (6.9)	16 (8.0)	4.0	5.7–333.0	<.001
Total	291	201			
T wave					
Biphasic: Pos-neg	2 (0.7)	0 (0)	Excluded from analysis		
Biphasic: Neg-pos	198 (67.3)	149 (50.7)	Ref.		
Monophasic: Pos	49 (16.7)	122 (41.5)	9.2	5.1–16.5	<.001
Monophasic: Neg	45 (15.3)	23 (7.8)	0.2	0.1–0.5	<.001
Total	294	294			

APD, atrial premature depolarization; OR, odds ration; CI, confidence interval.
Horse effect was significant for P wave morphology ($P < .001$) not for T wave morphology.

not change. The APDs were significantly associated with singular positive P waves and were more likely to have a single positive T wave.

In this study, only conducted APDs for which, the RR interval was at least 20% shorter than the RR interval of the preceding sinus beat were included. This value is probably relatively high, but was arbitrarily chosen because currently no data are available for differentiation between APDs and sinus arrhythmia.

On visual inspection, the most prominent feature of an APD compared to its preceding sinus beat was a more negative S wave and a singular, instead of bifid, P wave. Furthermore, T waves often changed polarity in the case of an APD. Another important finding in this study was that horse was a strongly significant factor for all observations. This was especially clear for the change in S wave amplitude. Among the different horses, the mean increase in S wave amplitude for an APD ranged from 3 ± 5 to $44 \pm 10\%$.

Overall, a significant negative relationship between RR interval and the relative increase in S wave amplitude was found, meaning that the shorter the RR coupling interval, the larger the S wave amplitude of the APD, when compared with the preceding sinus beat. This relationship, however, was strongly horse dependent and can be influenced by differences in vagal tone, intraventricular conduction and altered QRS depolarization or repolarization.[9] The RR interval also influenced T wave morphology. Those APDs with short RR interval were more likely to have monophasic positive T waves.

Current veterinary literature about ECG in horses usually states that, in the case of an APD, P wave morphology changes, whereas QRS and T wave morphology remains similar.[1,3,10] In our study, P wave morphology did indeed change. The APDs were more likely to have singular positive P waves, instead of the bifid P waves which were seen in most normal sinus beats. In the case of an APD, the impulse originates from another location and conducts differently over the atrial myocardium, which might lead to altered ECG deflections.[1,11] In our study, ECG registration was performed in a modified base-apex configuration because this configuration frequently is used in equine practice, especially during exercise and 24-hour ECG recording. This modified base-apex configuration produces fewer motion artifacts, but lower P waves compared with a true base-apex configuration in which the right arm electrode is placed in the jugular groove.[8] Furthermore, although in our study, all ECG measurements were performed using the second lead, the P wave in our electrode configuration generally was best visualized in the first lead of the ECG [between the right arm (−) and the left arm (+) electrode]. Those P waves buried in preceding T waves were therefore sometimes difficult to identify and could not be measured in 32% of the APDs. In these cases, leads 1 and 3 were very helpful to identify the APD. Inspecting multiple lead traces is therefore useful for ECG interpretation.

Although it usually is stated that in the case of APDs, ventricular conduction is not affected and QRS complex and T wave morphology do not change, our study shows that, in addition to changes in P wave morphology, APDs also can lead to changes in QRS complex and T wave morphology. The APDs resulted in PQ shortening, increased S wave amplitude and a more positive T wave. Similar changes occur during exercise because of a positive inotropic effect and an increase in sympathetic tone.[12,13]

Shortly coupled APDs may lead to very premature QRS complexes. For such short RR intervals, especially when occurring after a relatively long RR interval, the conduction system or parts of it might not be fully recovered yet, resulting in intraventricular conduction block. In other species, this conduction block commonly occurs at the right bundle branch and is known as Ashman phenomenon. The conduction block results in an aberrant conduction whereby part of the myocardium is depolarized from cell to cell. The ECG typically shows QRS widening with changes in QRS and T wave morphology. Whether similar mechanisms occur in horses is not known, but QRS widening did not occur. The increase in S amplitude and the T wave change toward a positive deflection are very similar to what occurs when heart rate physiologically increases (eg, during exercise). Indeed, in 13% of the horses we found

that, for the APDs, the RR interval and the change in S wave amplitude were inversely correlated, meaning that the shorter the RR coupling interval, the larger the difference in S wave amplitude between an APD and a sinus beat. In 83% of the horses, the relationship did not reach significance but this might be related to the small number of APDs (n = 10) per horse. Only in 1 horse was a positive relationship found. Also, T wave morphology was significantly influenced by changes in RR interval with a shorter RR interval leading to a more positive T wave.

A decrease in left ventricular preload has been shown to result in smaller QRS amplitudes, as explained by the Brody theory.[14,15] Premature depolarization results in a shorter diastolic interval and consequently a lower left ventricular preload. The increase in S wave amplitude found in our study can therefore not be explained by the Brody theory.

Respiration also influences ECG amplitudes because of alterations in transthoracic impedance and changes in cardiac filling.[16,17] However, this effect is not associated with APD occurrence and therefore not likely to have any effect on our results.

We chose not to include horses with VPDs, because in these horses one might argue whether an altered QRS morphology is a result of fusion beats. In horses with only APDs, this was very unlikely to occur.

In our study, a modified base-apex lead was used for ECG measurements because this lead often is used in clinical situations. The other Einthoven leads also were displayed for better interpretation of the ECG. However, one should be aware that most ECG units, such as the Televet100, only record lead 1 and 2 whereas lead 3 is calculated assuming an Einthoven triangle with the heart in the middle. In horses, these criteria are not fully met which results in minor changes, especially in amplitude. Nevertheless, using multiple leads can be helpful for ECG interpretation.[7]

This study has several limitations. Observers were not blinded to the electrocardiographic complexes, so they were aware of whether a cardiac cycle was a normal sinus beat or an APD. Blinding the observers would have been difficult to accomplish, because, for measuring the RR interval, the previous cycle must be included. As mentioned before, larger P waves could have been recorded from a true base-apex ECG. However, the placement of electrodes and lead recording were chosen to mimic the clinical situation. Furthermore, there was no control group of healthy horses included. A control group of horses without APDs would have allowed us to determine the natural variability in electrocardiographic measurements between successive normal sinus beats to compare those results with the variability between an APD and its preceding normal sinus beat. In our study, an APD was defined as an individual atrial depolarization with an RR coupling interval at least 20% shorter (arbitrary value) than the RR interval of the preceding sinus beat. We chose this relatively large difference as a criterion to minimize inclusion of early sinus beats. There is, however, a chance that some early sinus beats were included as APDs.

In conclusion, our study demonstrated that APDs can lead to important changes in ventricular morphology. Clinically, the most important observation is the fact that the S wave becomes more negative and the T wave often changes polarity from negative or biphasic to a monophasic positive T wave. These changes, which are strongly horse dependent, may lead to incorrect diagnoses, particularly when P waves are difficult to identify on the ECG. Clinicians should be aware of a possible change in QRS complex and T wave morphology not only with VPDs but also in the case of an APD to avoid mistakes. The use of multiple lead recordings can be useful to diagnose certain cardiac arrhythmias.[7]

Footnotes

[a] Televet100, Engel Engineering Services GmbH, Heusenstamm, Germany
[b] Mainat Vet, Barcelona, Spain
[c] Televet100 software version 5.1.2, Engel Engineering Services GmbH
[d] SAS Institute Inc., Cary, NC

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Conflict of Interest Declaration: Authors declare no conflict of interest.

Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

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